ease. In this chapter (on the nervous symptoms of fever), the whole question of uræmia is fully discussed.

Special.—The special objections to Dr. Mahomed's theory may be noted as follows.

Granting the existence of minute capillary hæmorrhages (which has yet to be demonstrated), and granting their competence to produce convulsions, how do they act? There are only two possible ways: (*a*) by loss of blood, and (*b*) by physical pressure on the cerebral substance (for such minute hæmorrhages could not lacerate the brain-tissue as do larger ones). But the hæmorrhages, as described by Dr. Mahomed, are so minute that the loss of blood cannot be felt by the brain. They must, therefore, act by compressing the brain-tissue. Now, if one or two minute hæmorrhagic points can produce convulsions in this way, we ought frequently to find such symptoms in those cases, first described by Charcot and Bouchard, in which minute capillary aneurisms exist in large numbers in the brain, producing, in virtue of their number, greater pressure than could be caused by a few hæmorrhages not exceeding in size the individual aneurisms. But such is not the case. If Dr. Mahomed will refer to the published observations of the French pathologists, he will find that miliary aneurisms, which differ from his hæmorrhages only in having the blood surrounded by the aneurismal covering, may exist in the brain in dozens, and even in hundreds, without causing any such symptoms as those which he attributes to minute capillary hæmorrhage. A certain allowance, I am aware, must be made for the more sudden occurrence of the hæmorrhagic pressure; but this I believe to be more than counterbalanced by the large number of the aneurisms. These aneurisms, too, occur in all parts of the brain: the optic thalami, the corpora striata, the convolutions, the pons Varolii, the cerebellum, the centrum ovale, the middle peduncles of the cerebellum, the peduncles of the brain, and the bulb.

To sum up: the objections to Dr. Mahomed's views are, first, that they lay aside as inadequate an agency which is at work in every case of uræmia, and which has been proved to be a potent cause of convulsions—viz., malnutrition of the nervous centres; and, second, that they substitute for this an agency whose existence has not been proved, and whose competence to produce convulsions is, at the least, doubtful.

ON A CASE OF DOUBLE HEMIPLEGIA, WITH CEREBRAL SYMMETRICAL LESIONS.

By THOMAS BARLOW, M.D., B.S.,
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W. S., A BOY aged 10, was admitted into the Hospital for Sick Children, Great Ormond Street, under the care of Dr. Dickinson, on December 16th, 1875. There was no history of rheumatism, and no suspicion on the part of his mother that the boy had heart-disease.

Four months before admission, according to the statements given, he must have had an attack of right hemiplegia. Whilst holding the baby, he suddenly burst out crying, his face was drawn, and he fell down. But he did not lose his senses, and within a quarter of an hour was able to walk. He was put to bed, and at that time he talked naturally and swallowed without trouble. But next morning, he had lost his speech; he could only say "Haw-haw"; he had also difficulty in swallowing. His face was drawn. He could not grasp with the right hand, and he dragged the right leg when put on his feet. In ten days, he was greatly improved. The leg improved before the arm. The speech had returned on the tenth day; but occasionally he made a mistake, gave the wrong name for a boy, and did not seem always quite to understand what was said to him.

In a month's time, he seems to have quite recovered. He was then able to go errands for his mother, and he discharged them correctly. He was very pale. Six days before admission, he got an attack of left hemiplegia. It was noticed that he was very irritable, and could not bear the noise made by the other children; he cried and sobbed a great deal. Next morning, he could not use the left hand, had difficulty in swallowing, and had quite lost his speech. His mother does not know whether the face was drawn or not. No notice was taken of his legs till five days afterwards, and then it was found that he could stand without support. When brought to the hospital, he cried just like a patient with senile softening, directly he was approached. But after a few days, he was amenable to investigation. As he changed very little, I may summarise his condition whilst in the hospital.

The only approach he could make to a voluntary articulate sound was "Ah". He could cry vigorously; there was no lack of voice. From his admission, he appeared to understand all that was said to him. When asked his age, he counted ten on the questioner's fingers; counted four, when asked how many brothers he had; and so forth. He made signs for what he wanted, and the signs were correct.

From the first, he was able to write his name when asked, and after a few weeks would answer in writing any question that was put to him.

He could not be induced to show his upper teeth, nor to protrude his lips, nor to smile when he was told. But his face was not expressionless. He occasionally smiled involuntarily. There was at first the slightest possible flattening of the left naso-oral ridge, but this soon passed off, and there was no loss of symmetry.

He could not protrude his tongue at all. There was no wasting of the tongue. Both sides responded to Faradism. There was no deviation of the uvula; no dropping of either palatine arch; no regurgitation of fluids. The palate was sensitive. His swallowing of solids was very peculiar. He was fed best when lying on his back. A large morsel succeeded better than a small one. It seemed very gradually to find its way to the fauces, but without any arching up of the tongue. When once past the arch of the palate, there was at no time any difficulty. There was no choking nor regurgitation; nor did the boy push the morsel down to the back of the pharynx, as is seen sometimes in cases of labio-glosso-pharyngeal paralysis.

He never made any attempt to chew. He did not use his pterygoids at all; but on one occasion, after much persuasion, he was induced to give my finger a bite between his molars, and the masseters were felt to contract. The sensory division of the fifth nerve was quite natural. There was no ocular palsy; the optic discs were natural. The sense of taste and the sense of smell were natural.

To sum up the cerebral condition, there appeared to be loss of voluntary motor power over the muscles concerned in articulation and the first part of deglutition.

As to the limbs, there was at first weak grasp with both hands, the left the weaker. There was slight wasting of both upper limbs. But all this greatly improved in three weeks' time. At no time was any rigidity elicited by flexion. Both arms responded well to Faradism. At the time of his admission, he was able to draw up his lower limbs equally; and in a month, when he was allowed to walk, he did so without dragging. There was no diminution of irritability to the Faradic current.

There was no perversion of sensation. For a couple of days after admission, he had retention of urine, but this did not recur. There was no trouble with the sphincter ani. The cause of his hemiplegia was not far to seek. He had a double aortic murmur, with considerable hypertrophy and dilatation of the heart.

He was in the hospital less than two months. His condition as to speech and swallowing did not alter in the least, though his limbs improved. No fresh nervous signs appeared.

He died from the results of his aortic regurgitation. At the *post mortem* examination, the right and left aortic valves together measured the same width as the posterior aortic valve. They were partly united along one edge, and there was a frænum uniting two contiguous cusps. This was probably a congenital condition. Besides this, there were vegetations partly calcified at the free margins of all three valves, and there were a few recent vegetations at the base of the aortic curtain of the mitral. There was dilatation and hypertrophy; and there were some oldish thrombi in both auricles. As to the other viscera, there was some induration with old infarcts in the lungs, a nutmeg liver, infarcts in the spleen and kidneys, and some effusion, probably passive, in the peritoneum.

As to the brain, no disease of the vessels forming the circle of Willis could be detected; but in the left middle cerebral, an inch and a quarter beyond its origin, three calcified nodules, each rather less than a millet-seed, were found. Two branches, traced from this vessel, were also found to present calcified spots. There was no actual occlusion, but the calibre was obviously diminished by the above condition. No aneurism was detected and no plug of fibrine found. In an identically corresponding position on the right side some similar calcification was found, though not quite so much in amount. These vessels were traced into small oval regions of softening, each of which might be covered with a shilling. On each side, these regions consisted of the lower end of the ascending frontal and the hinder end of the middle and inferior frontal convolutions. These areas were pale, buff-coloured, slightly depressed, slightly softer than the surrounding brain-substance. Reckoning from the surface, they were less than a quarter of an inch deep, *i.e.*, they involved the cortical and a little of the subjacent white substance.

The rest of the brain, so far as could be made out by naked-eye inspection, was natural.

REMARKS.—The first point of interest as to this case is its etiology. The very close union of two of the aortic valves appears to me strongly in favour of a congenital condition of imperfect development. Whether there was fœtal endocarditis, I will not pretend to say; but it seems likely that the congenital imperfection predisposed to the subsequent endocarditis in this region.

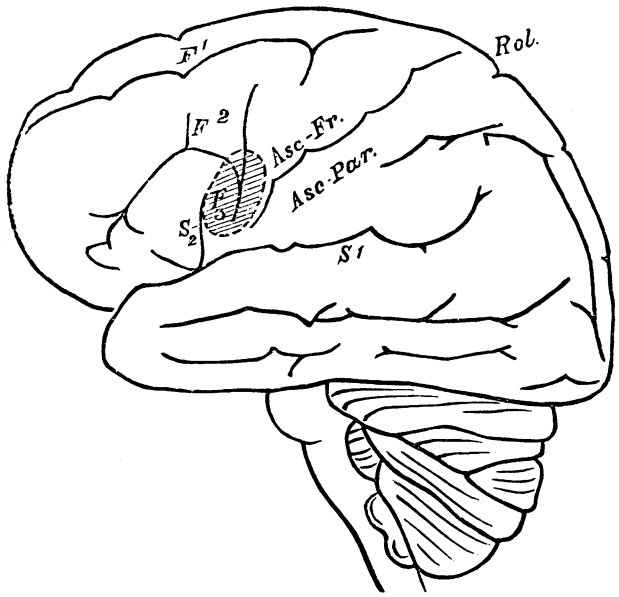

Fig. 1.—Lateral View of Human Brain (Ecker). S_1. Posterior Limb of Fissure of Sylvius. S_2. Anterior Limb of Fissure of Sylvius. Rol. Fissure of Rolando. Asc. Fr. Ascending Frontal Convolution. Asc. Par. Ascending Parietal Convolution. F_1, F_2, F_3. Superior Middle and Inferior Frontal Convolutions. The dotted portion represents the situation of the lesion on each side of the brain—viz., in the lower end of the ascending frontal and the hinder end of the middle and inferior frontal convolutions.

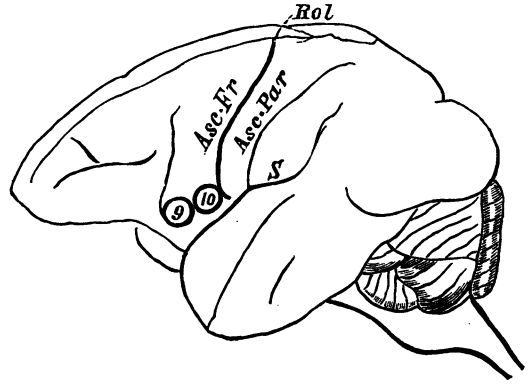

Fig. 2.—Lateral View of Brain of Monkey (adapted from Ferrier). Stimulation over areas (9) and (10) produced movements of mouth and tongue. These areas correspond with the lower portion of the ascending frontal and the hinder end of the inferior frontal in man.

The sequence of events seems clear, viz., that vegetations were carried either from these valves or from the left auricle to the spleen and kidneys, and also, first, to the branches of the left middle cerebral, and afterwards to the branches of the right middle cerebral. There, these vegetations first caused block, and subsequently set up changes in the walls of the vessels and in the parts of the brain supplied by them.

It seems fair to associate each attack of hemiplegia with diminished blood-supply, first to the left, afterwards to the right, side of the brain, in the convolutions round the fissure of Rolando. But, whilst the greater part of the regions referred to recovered, the nutrition of one small portion on each side was *permanently damaged.*

Is it unreasonable to associate this bilateral lesion with the only marked symptom which remained after the two attacks of hemiplegia had passed off, viz., the loss of voluntary power over the mouth and tongue muscles? I own that, at first, the question of disease of the pons or medulla came into my mind; but the entire absence of wasting of the tongue, the absence of ocular palsy, the non-implication of the sensory division of the fifth nerve, and the very limited affection of the facial, were surely strong points against such a condition. Dr. Hughlings Jackson, who was kind enough to look at the boy, predicted disease of the regions supplied by branches of the two middle cerebrals.

The relation between the specialised symptoms during life and the well-defined lesions found *post mortem*, is substantiated by the results of physiological experiment. Professor Ferrier has shown, in the brains of monkeys, that in this identical region, or rather in the region homologous with it, the centre for the movements of the mouth and tongue is situated. Now, according to Dr. Broadbent's hypothesis, the bilateral muscles, which act together, are represented on the two sides of the brain. After the first attack of hemiplegia, although this region on the left side was probably permanently damaged, yet still the right side remained intact. But, after the second attack of hemiplegia, the corresponding region on the right side became damaged; and henceforth, as far as voluntary movements of the mouth and tongue were concerned, the boy was irretrievably deficient.

The case is incomplete, because of the absence of a microscopic examination of the pons Varolii. The brain was put into spirit, but did not harden properly. The lower part of the medulla and cervical region of the cord, however, were preserved; and I have to thank my friend Mr. R. W. Parker for some sections of them. In the spinal cord sections, there is undoubtedly some thickening of the neuroglia generally. It is most marked in the front of the posterior columns. What the significance of it is, I am not prepared to say. It is quite different from the descending sclerosis described by Charcot. In the medulla oblongata, Dr. Gowers (who has been good enough to look over the sections) has failed to find anything wrong, except that the cells of the nuclei of the vagi seem a little smaller than natural.

ON THE TREATMENT OF ACUTE RHEUMATISM.*

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DR. LATHAM says: "Acute rheumatism has experienced strange things at the hands of medical men. No disease has been treated by such various and opposite methods. Venesection has wrought its cure, and so has opium, and so has calomel, and so has colchicum, and so have drastic purgatives. I speak of each of these remedies in the sense which medical men imply when they talk (as they often do) of this, that, or the other thing being their 'sheet-anchor'—meaning that they rest upon it alone for the cure of the rheumatism, and employ other remedies either not at all or for very subordinate purposes. And, indeed, I bear my testimony to the success of each of these different remedies so far as that, under the use of each, I have seen patients *get well.*"

Had he written in these days, Dr. Latham would have had many other "strange things" to add to his list: alkalies, acids, salines, hot water, cold water, lemon-juice, citric acid, chloral, belladonna, iodide of potassium, ergot, digitalis, aconite, guaiacum, emetics, sulphur, antimony, perchloride of iron, quinine, iodine, plaster of Paris bandages, galvanism, subcutaneous injection of carbolic acid, blisters, podophyllum, cynara, propylamine, chloro-hydrate of trimethylamine, and last, but not least, salicin, salicylic acid, and salicylate of soda. All these remedies have been used for rheumatic fever, and each has been extolled as more potent than all the rest.

It is, I take it, the duty of the opener of the debate to facilitate discussion by briefly laying before you the results of past experience. I will, therefore, give a summary of the effects of the more important of the above remedies.

First, however, arises the question, What are the *natural* course and duration of acute rheumatism? We all know that rheumatic fever has no definite course, and is most uncertain in its duration; that some cases will be long protracted, others will be well in two or three days; hence our difficulty in deciding on the merits of the various methods of treatment. The results of the *mint-water* treatment by Drs. Gull and Sutton drew special attention to the fact that rapid recoveries were often made when the only treatment was good nursing, and that

* Read before the Bath and Bristol Branch: being the opening paper at a discussion on the Treatment of Acute Rheumatism.